

Case Reports

Occupational asthma due to chloramine-T solution

V. M. KUJALA*[‡], K. E. REIJULA*, E-M. RUOTSALAINEN[†] AND K. HEIKKINEN[†]

**Department of Occupational Medicine, Oulu University Hospital, P.O. Box 22, FIN-90220 Oulu, and*

†Department of Pulmonology, Päivärinne Hospital, FIN-91410 Jokirinne, Finland

Introduction

Chloramine-T, a potent disinfectant has been used to clean surfaces in butcheries, kitchens, operating rooms, laboratories and dairies. In 1945, Feinburg and Watrous (1) found occupational asthma and rhinitis in workers exposed to chloramine-T. The diagnosis was based on wheal and flare skin reactions to chloramine-T. So far, chloramine-T allergy has only been described in subjects who have been in contact with the powder of the disinfectant. To our knowledge, this report is the first to describe the provocation of occupational asthma by aerogenic exposure to commercial chloramine-T solution.

Case Report

A 36-year-old non-atopic female cleaner who had never smoked regularly presented with sneezing, bronchial coughing and dyspnoea a few months after she started to use a new disinfectant at work. The respiratory symptoms appeared immediately after the exposure to chloramine-T and continued several hours after the working shift. The symptoms disappeared when she had to stay out of work.

At work, she cleaned showers and saunas at a municipal indoor swimming pool. Chloramine-T (Alinex[®]) was mainly used in a 10% solution (10 mg ml⁻¹) and the worker sprayed the disinfectant on the walls and floors with pressurized water. The temperature in the working environment was 30°C and the relative humidity of the tidal air was 50–80% although there was mechanical extract ventilation in the room and the extracted air rate was

up to 6.2 dm³ s⁻¹ m⁻² of the floor. PEF values decreased from baseline 470 l min⁻¹ to 400 l min⁻¹ (15%) at work.

In the clinical examination, chest auscultation was normal. Leucocyte count was 4.500 with 2.5% eosinophils. Total serum IgE was 151 IU l⁻¹ and the specific IgE to chloramine-T was 16.27 IU l⁻¹ (Pharmacia Diagnostics, Uppsala, Sweden). Skin prick tests with common inhalant allergens were negative. Skin prick test with chloramine-T solution (0.5 mg ml⁻¹) showed an immediate wheal and flare reaction which was as large as the reaction caused by 1% histamine dihydrochloride (used as a positive control). Chest radiograph was normal. In lung function tests, forced vital capacity (FVC) was 4.43 l [109% of predictive value (2)], FEV₁ was 3.15 l (91%) and FEV% was 71%. Histamine provocation test revealed symptoms of moderate bronchial hyper-reactivity, and the provocative dose of histamine diphosphate which induced a 15% fall in FEV₁ was 0.18 mg.

Bronchial provocation was performed by using a Spira Elektro 2[®] dosimeter (3) (Hengitys-hoitokeskus, Hämeenlinna, Finland). First the patient inhaled saline as placebo which did not significantly change PEF values over 24 h. The chloramine provocation was performed using a single inhalation of chloramine-T solution (0.5 mg ml⁻¹) with a Spira Elektro 2[®] dosimeter. In this automatic inhalation-synchronized dosimeter, aerosol delivery time was adjusted to 0.2 s and the threshold volume of inspiration to 100 ml. The provocative dose of chloramine-T was 2.0 µg. After 15 min, the patient developed rhinorrhea, coughing, dyspnoea and bronchial wheezes. In spirometry (Fig. 1), FEV₁ decreased from 3.05 l to 1.65 l (46%) and PEF values decreased from 475 l min⁻¹ to 295 l min⁻¹ (38%). Symptoms disappeared and PEF values returned to normal within 3 h of administration of inhaled broncho-

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[‡]Author to whom correspondence should be addressed at: Department of Public Health Science and General Practice, University of Oulu, Aapistie 1, FIN-90220 Oulu, Finland.

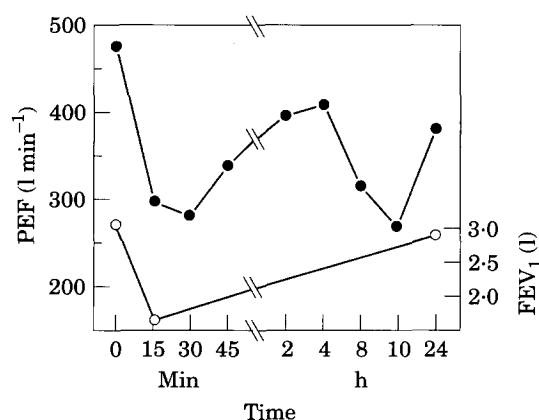


Fig. 1 The results of pulmonic functions (PEF and FEV₁) after one inhalation of chloramine-T solution (0.5 mg ml⁻¹) with Spira Elektro 2[®] dosimeter. ●, PEF; ○, FEV₁.

dilator. Between 4–10 h after inhalation, the patient experienced dyspnoea again, accompanied by wheezing. A fall in PEF values of 43% of the pre-inhalation value reoccurred. FEV₁ was not measured during the late reaction. Arterial blood oxygen pressure was decreased to 7.85 kPa. Pulmonary diffusing capacity was normal and chest radiograph showed no abnormalities. Leucocyte and eosinophil counts in blood remained normal.

A diagnosis of chloramine-T-induced occupational asthma was confirmed on the basis of positive skin prick test, RAST, bronchial provocation test results, adequate workplace exposure, and onset of work-related asthmatic symptoms.

Discussion

Chloramine-T is a water soluble powder with potent oxidizing, anti-viral, bactericidal and fungicidal properties. A chloramine-T powder has previously been reported to cause occupational asthma (1,4,6,7). In 1979, Bourne *et al.* (4) provided evidence that these asthmatic symptoms were of allergic origin and soon after, Kramps *et al.* (5) demonstrated that the asthmatic reaction was due to specific IgE-mediated reactions caused by chloramine-T. In 1981, Dijkman *et al.* (6) were able to show that the bronchial obstruction due to chloramine-T can be either an immediate (develops 10–20 min after exposure) or late onset type of reaction (develops 4–8 h after exposure). The antigenic determinant responsible for the immunological reaction has been suggested to be formed by the para-toluenesulfonyl group of the chloramine-T molecule (5).

In this case report, the previously healthy cleaner developed an immediate and late bronchial obstruction after the exposure to chloramine-T solution. She had asthmatic symptoms during and after the working shift. No bronchial wheezing on chest auscultation was detected during the first clinical examination. After the provocation test, however, the patient developed severe dyspnoea the duration of which was longer than was expected. The dose of chloramine-T used in the provocation test was selected according to previous studies (6) and the solution was inhaled by Spira Elektro 2[®] dosimeter. In addition, the chloramine-T concentration used in the inhalation challenge test was assumed to correspond to the chloramine-T exposure at the working place. According to our experience in this case, however, the provocation test should be started with a solution containing 0.05 mg ml⁻¹ of chloramine-T and if an immediate reaction does not occur, provocation could be continued with a solution of 0.5 mg ml⁻¹.

In earlier studies, specific IgE antibodies to chloramine-T have been detected in patients' sera. On the contrary, specific IgG antibody isotypes have not been associated with chloramine-T allergies (5). In this report, the patient had an increased total serum IgE and specific IgE to chloramine-T.

The patient in this case report had respiratory symptoms soon after she started to use chloramine-T solution as a disinfectant. The working environment where she did the cleaning was equipped with a mechanical extract air ventilation system. The artificial ventilation was insufficient to impede the hazardous increase in the concentration of the aerosolized and vaporized allergen in the room air. Especially at the time of spraying, with the allergenic solution in humid and warm working conditions, the health hazard is obvious. It is unknown what the sufficient air change rate is in purpose to avoid sensitization during disinfection in special environments. A non-sensitizing solution should be used in such cleaning work to avoid allergic disorders.

In conclusion, chloramine-T which is used as a potent disinfectant may cause a health hazard to cleaners who are exposed to chloramine-T aerosol. After aerogenic exposure to chloramine-T solution, the risk of developing hypersensitivity reactions, rhinitis and bronchial asthma is increased.

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